Abstract

Physiological dormancy is an important developmental trait ensuring that seed does not germinate when the environmental factors are appropriate only temporary. The transition from seed dormancy to germination is regulated by a large number of factors and the phytohormone abscisic acid (ABA) plays a crucial role. Enhanced response to ABA and its biosynthesis is a key mechanism in dormancy induction and maintenance. ABA interacts antagonistically with gibberellins (GAs). Therefore GA biosynthesis and ABA catabolism are positive germination regulators. However, other phytohormones are also involved in the regulation of dormancy and germination. The most studied is ethylene which supports germination similarly to GA. Numerous factors affect dormancy at molecular level, namely chromatin remodeling, gene products that function only in dormancy regulation [e.g. *DELAY OF GERMINATION 1* (*DOG1*)] or gene products that mediate seed response to environmental factors. The dormancy, its induction, depth and release, is driven not only by environmental conditions affecting mature seeds, but also by conditions acting during seed maturation in a maternal plant when the primary dormancy is induced. Requirements for dormancy release and germination induction may vary considerably between species. The physiological dormancy is therefore a complexly controlled process which is affected by a large number of factors.